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THE MECHANICS OF BREATHING FOLLOWING

OPEN HEART SURGERY

by

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A THESIS

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies for acceptance, a thesis entitled "The Mechanics of Breathing Following Open Heart Surgery", submitted by Arthur R. Macneil in partial fulfillment of the requirements for the Degree of Master of Science (Medicine).

ABSTRACT

The mechanics of breathing; i.e. compliance, airflow resistance and the work of breathing were determined in nineteen patients scheduled for open heart surgery utilizing cardiopulmonary bypass. During the first day of the postoperative period the measurements were repeated.

The lung compliance was found to decrease. This decrease is thought attributable to mechanical factors rather than to an actual change in the pulmonary parenchyma. The expiratory airflow resistance increased in the postoperative period. Although no definite explanation is given, several possible causes of this phenomenon are discussed.

The changes in the various fractions of the work of breathing tended to parallel their analogues of compliance and airflow resistance. The net effect was a significant increase in the work of breathing.

TABLE OF CONTENTS

	Page
Introduction	1
Review of the Literature	2
Methods	13
Apparatus and Technique	13
Procedure	25
Analysis of the Records	26
Techniques	30
Results	38
Discussion	51
Conclusion	59
Bibliography	
Appendix I	Case Summaries
Appendix II	Normal Subjects
Appendix III	Effect of Oesophageal Balloon Being Attached to Nasogastric Tube
Appendix IV	Calibration of Wedge Spirometer
Appendix V	Calibration of Polar Planimeter
Appendix VI	Abbreviations

LIST OF FIGURES

			Page
Figure	1	Godart Pulmotest	14
Figure	2	Oesophageal Balloon	18
Figure	3	Oesophageal Balloon with Levin Tube	19
Figure	4	Wedge Spirometer	21
Figure	5	Diagram of Work Loop	31
Figure	6	Sample Tracing	37
Figure	7		Appendix V

INTRODUCTION

In the decade since the advent of the cardiopulmonary bypass man has been able at last, to treat definitively, many types of cardiac lesions, both congenital and acquired. Early in the experience of all centers was noted a high incidence of post-operative respiratory insufficiency. This has constituted a major cause of death in these patients.⁵

The majority of patients dying in respiratory failure had suffered the "Post Perfusion Lung Syndrome" described at some length herein. It is well to explain the various factors involved in this syndrome, since all of these phenomena could alter pulmonary function in the postoperative period.

At the time this study was started there was virtually no adequate study of the mechanics of breathing, in the immediate postoperative period, reported in the literature. This work was undertaken to document which changes did occur.

REVIEW OF THE LITERATURE

The Morbid State.

Rasky and Rodman⁴⁷ cite two types of respiratory distress following open heart surgery. First, is insufficiency of respiratory gas exchange. They tend to regard this as a non specific phenomenon with many factors contributing to its pathogenesis; principally increased metabolic demands, sedation, trauma to the chest, secretions in the tracheo-bronchial tree and incisional pain. All these are reasonable causes for limitation of ventilation. However, the authors go on to mention other factors, such as, loss of surfactant and resulting atelectasis, intrapulmonary hemorrhage secondary to clotting disorder, and increased pulmonary capillary permeability. In citing these last factors the author fails to make an adequate distinction between the above and the second type of respiratory distress seen in the postoperative period;- the Post Perfusion Lung Syndrome.

The Post Perfusion Lung Syndrome was described by Dodrill¹⁵ as follows:

"Immediately after the conclusion of the operation the respiratory rate of the patient is increased. It is often 40-50 per minute. There may be slight cyanosis. The condition may become progressively worse and death usually occurs on the second or third day, although the lives of some patients may be prolonged for six to seven days. There are signs of increasing pulmonary insufficiency as time goes on. The heart and blood pressure remain good until the very end. It is primarily a pulmonary death. With less severe injuries the patient gradually recovers."

Baer and Osborn⁵ made note that hypotension may occur in the morbid picture; and further emphasize the progressive cyanosis and include fever as part of the clinical presentation. They went on to describe the pathology of the diseased lungs:

"Autopsy findings on patients who have died with this syndrome are depressingly similar, both from patient to patient and from institute to institute..... Seventy-two percent of the 41 patients who died following perfusion at Stanford Hospital between March 1957 and May 1959 manifested the perfusion syndrome. Postmortem studies revealed that their lungs were dark red and congested, with focal zones of collapse and parenchymal hemorrhages. The hemorrhages varied in size from 1 mm. to several centimeters in diameter. In most instances, they were observed subpleurally and throughout the deep parenchyma, in all portions of both lungs. Blood was frequently present within the bronchi. The remainder of the organs were not remarkable, except for generalized hyperemia.

Nahas et al⁴¹ describes a similar histological picture in the lungs of dogs perfused with homologous blood and noted the appearance when methyl-green-pyronin stains were used, the presence around the alveoli of an abnormal concentration of large cells with irregular pyroninophilic cytoplasm and large rounded nuclei resembling the immature plasma cell. In a following report Nahas⁴² emphasizes that these cells are in close resemblance to those seen infiltrating a homotransplanted heart or kidney. He further emphasized that these cells are not found when homologous blood is not used in the perfusion.

Pathogenesis.

The pathogenesis of the syndrome remains an enigma. Among the earliest writers advancing speculations were Kolff et al²⁵

and Dodrill¹⁵ who emphasized mechanical and chemical factors. Kolff et al²⁵ observed that the syndrome was twice as common in those patients with pre-existing pulmonary vascular disease and more common in small children. As one possible factor they cite dessication of the lungs due to keeping them inflated with dry oxygen. The possibility of damage due to lowering the colloid osmotic pressure of the blood by excessive dilutions with electrolyte solutions was advanced. Air embolism was also mentioned as a possible factor. The writers however advanced as their principal hypothesis; that the cause of the post perfusion lung syndrome was temporary overloading of the pulmonary circulation. This might take place by any of several mechanisms.

1. By forward overfilling

- a. During partial by-pass if a sudden increase in the patient's blood volume occurs.
- b. During total by-pass by accumulation of extravagant coronary venous blood in the right heart.

2. Through collateral vessels

- a. Open patent ductus arteriosus
- b. Increased bronchial circulation

3. By retrograde overfilling

- a. By mechanical factors
 - i. Obstruction of pulmonary venous return
 - ii. Mitral stenosis
 - iii. Aortic and mitral regurgitation

b. Dynamic impediment (inability of the left ventricle to overcome the aortic pressure).

i. Ventricular fibrillation

ii. Ineffective left ventricular beat.

Awad et al,⁴ in an exhaustive review cite as a possible factor in addition to those mentioned above, the possibility of low perfusing pressures being responsible for an increased tendency to sludging and a decreased suspension stability of the erythrocytes, thus resulting in a slowing of the microcirculation and pulmonary congestion. This produces a pathologic picture strikingly similar to that seen in shock and many non specific varieties of trauma, as cited by Baer and Osborn.⁵ Awad et al⁴ also cite anoxia as a possible factor in the etiology. This now is supported in part by Williams et al. ⁵⁶

In the earlier years of open heart surgery the mechanical factors were thought to be the prime cause of the syndrome. As early as 1958 however, more subtle thoughts were advanced. At that time Bahanson⁶ made the suggestion that incomplete cleaning of the perfusion apparatus might be an operative factor and made note that all pulmonary complications were less frequently seen with the use of completely disposable equipment or completely clean components. Baer and Osborn in 1960⁵ produced experimental evidence that simple denaturation of the blood due to turbulence could cause a similar histological picture even if the chest of the experimental animal were untouched.

Lee and his co-workers²⁷ supported this concept of trauma to the blood particularly emphasizing the role of denaturation of plasma proteins. Neville et al⁴³ cited destruction of the red cells as causative of pulmonary vasculitis, and inflammatory response.

As perhaps secondary changes, Tooley et al⁵¹ in 1961 and later, Mandelbaum and Giammona³⁰ in 1964 give evidence that there is a decrease in pulmonary surfactant in the lungs following by-pass: However they could isolate from the pump blood no substance inhibiting pulmonary surfactant. Trimble⁵² and associates have suggested that heparin which activates lipoprotein lipase in blood might be responsible for the destruction of the lipoprotein surfactant during perfusion. (Heparin is used as an anticoagulant in cardiopulmonary bypass procedures.) Dissenting voices are raised by Pattle and Burgess,⁴⁶ and Sutnik et al⁵⁰ who hypothesize and present evidence that changes in surfactant may be secondary to, rather than causative of atelectasis.

Recently, the use of a homologous blood prime for the apparatus has been assigned a possible place in etiology of the syndrome. Workers in the field such as Gadboys et al²⁰ and Daly et al¹⁴ have documented changes in the lungs with the use of homologous blood. This is further supported by the work of Nahas et al^{41,42} who excluded one lung of a dog from the circulation and perfused the other and documented changes in the perfused lung. (see above pg.3) They concluded that there is a damaging

factor in the perfusion blood and that the use of non homologous blood would prevent the major portion of the syndrome, i.e. disordered ventilation perfusion ratios.

PATHOPHYSIOLOGY

Pathophysiology of the Postoperative State. Immediate Changes:

There is a distinct lack of information available concerning the changes in lung function in the immediate postoperative period.

In 1960, Howatt et al²⁴ studied eleven patients who underwent correction of congenital or acquired cardiac lesions with the aid of extracorporeal circulation. Measuring the vital capacity, tidal volume and minute ventilation, maximum breathing capacity, diffusion capacity and functional residual capacity preoperatively; he then reassessed all parameters for several days after operation. He found, that in the first postoperative day the vital capacity dropped to a mean of approximately 25% of the preoperative values. The tidal volume showed a similar decrease, and the functional residual capacity dropped to 71% of the previous recorded value. There was a slight increase in the residual volume. Minute ventilation was maintained at near preoperative values by a decrease in tidal volume and an increase in the respiratory rate. There was a uniform reduction in the diffusion capacity. The tendency was for the lung volumes and minute volume; with the exception of the inspiratory reserve volume, to return to the preoperative value by the eighth post-

operative day. The diffusion capacity was lowered on the first day, and tended to increase gradually, although the vast majority had not returned to their preoperative value when remeasured at three to eight months. The authors admit that this might be expected following the repair of congenital heart lesions with left to right shunting of blood, but could offer no explanation for the changes following the repair of left sided valvular lesions. The volume changes described are attributed by the authors to the limitation of respiratory excursions by the painful thoracic wound.

Hedley-Whyte et al²³ confined their studies to patients with left sided valvular lesions who underwent surgery: some with, and some without the use of cardiopulmonary bypass. The main parameters assessed were those measurements giving data on ventilation and perfusion. They found large physiological shunts leading to arterial hypoxia in both groups. There was no significant difference between the magnitude of shunts of those patients who did, and those patients who did not have extracorporeal circulation; thus suggesting that the hypoxia is a non specific response to major surgery.

Weintraub et al⁵⁴ studied nine patients preoperatively and again approximately fifty days postoperatively. All had acquired valvular disease, and were subjected to cardiopulmonary bypass for definitive correction of their lesions. No significant change was found in the vital capacity, residual

volume, total lung capacity, functional residual volume, maximum voluntary ventilation or forced expiratory volume postoperatively. The mean shunt fraction of cardiac output increased significantly from 3.1% to 4.1% postoperatively.

No change was reported in the alveolar-arterial oxygen difference postoperatively. The non-shunt component of the alveolar arterial oxygen difference did not change postoperatively.

In 1966, Sullivan et al,⁴⁹ measured dynamic total respiratory compliance, and the work of breathing before and after cardiopulmonary bypass. It is unfortunate that their published data gives the work of breathing in kilogram centimeters and no attempt apparently was made to correct the work of breathing for variations in rate and depth, so that their published data on this parameter must be regarded as essentially useless. Their compliance measurements taken before and after cardiopulmonary bypass failed to show a significant change.

Mandelbaum and his co-workers³⁰ found no significant changes in compliance in his experimental animals after two hours of cardiopulmonary bypass. After four hours significant changes were observed. Cartwright et al¹⁰ also found significant change in compliance during extracorporeal circulation but these were related to methods of ventilation. Gustavino et al²² working with dogs, did describe a change during bypass but this was attributed to a technical failure in the bypass procedure, i.e. inadequate drainage of the left atrium.

Osborn et al^{44,45} attempted to study postoperative work of breathing but mentions only that no specific pattern was found.

LATER CHANGES:

Woolf and Aguzzi⁵⁶ describe their assessment of pulmonary function in 27 patients with left heart valvular lesions and 19 patients with septal defects before, and six to eight months following cardiac surgery done without cardiopulmonary bypass. They found significant decreases in the vital capacity in those patients with left heart valvular lesions postoperatively. The FEV was significantly decreased in the patients with left heart valvular lesions postoperatively, but not in those with septal defects. Neither the compliance, nor the resistance changed significantly in either group. The figures published for total work of breathing gave the work as kilogram meters per minute. Since there is no evidence of these being corrected for rate and depth of breathing, they must be regarded as meaningless.

Larmi and Appelquist²⁶ studied nine patients with mitral stenosis, eight patients with patent ductus arteriosus, one with concretio cordis and one with coarctation of the aorta, determining the compliance and inspiratory resistance preoperatively. Three weeks after surgery the measurements were repeated in four of the patients with mitral stenosis,

seven of those with patent ductus arteriosus and in the patients with coarctation of the aorta and concretio cordis.

Postoperatively the compliance values were unchanged or increased in three of the four patients with mitral stenosis, the remaining patient had a pulmonary infarction and hence the compliance decreased. In the patient with concretio cordis the compliance increased postoperatively. Two of the six patients with patent ductus arteriosus had an increase in compliance and it decreased slightly in four. In the patient with coarctation of the aorta the compliance decreased.

SUMMARY OF THE LITERATURE

Two types of respiratory distress following cardiac surgery have been described. The first type is a non specific respiratory depression caused by many non-specific factors such as incisional pain, narcotics, anaesthetic gases etc. The second type of distress is the Post Perfusion Lung Syndrome. This is a clinical picture consisting of dyspnea and cyanosis. The dyspnea is apparently on the basis of stiffened lungs and the cyanosis is attributed principally to the perfusion of unventilated alveoli is areas of atelectasis. The only specific pathological manifestation is the appearance of cells which resemble immature plasma cells. These appear to indicate an immunological element in the pathological picture. In the ten years that the syndrome has been described it was given a number of hypothetical causes. In the earlier

portions of the decade the tendency was to attribute it to mechanical factors such as overloading of the pulmonary circulatory bed, dessication of the lungs by dry oxygen, or air embolism. As these mechanical problems were gradually solved by technical advances, workers turned their attention to other and more subtle causative factors. It gradually became more apparent that some factor carried in the blood from the cardio-pulmonary bypass was capable of causing the changes of the post perfusion lung syndrome. The most notable suggestions have been that heparin used in the bypass procedure as an anti-coagulant might be leading to the inactivation of surfactant. The work of Nahas et al,^{41,42} Daly et al¹⁴ and Gadboys et al²⁰ suggesting that the use of homologous blood is responsible is impressive.

No definitive statement can yet be made as to the cause of the post perfusion lung syndrome. There are in all probability several factors operating. Current emphasis is being placed on the role of surfactant (or its lack) and the immunological reactions to whole blood. There is as yet no clue to a unified concept.

The functions of the lungs are disturbed in various ways in the post operative state. It is known that the lungs operate at a lowered volume in the immediate post operative period. The diffusion capacity is also lowered in this period. Alterations of ventilation-perfusion ratios in the post operative

period have been well documented.

The only change in mechanics of breathing described is that found in experimental animals by Mandelbaum and Giammona.³⁰ They found a decrease in compliance after prolonged bypass, and this was correlated with a decrease in surfactant.

At six months following surgery it was demonstrated that the vital capacity decreased in those with left heart lesions. The forced vital capacity was decreased in those with left heart lesions. No significant changes could be found in the mechanics of breathing.

METHODS

Selection of Patients:

Patients were selected at random from those scheduled for repair of cardiac defects using cardiopulmonary bypass. There were nineteen subjects. The ages of the subjects ranged from fourteen to sixty-three years.

No subjects younger than fourteen years of age were chosen as it is difficult to elicit their cooperation in the insertion of an esophageal balloon.

APPARATUS AND TECHNIQUE

Lung Volumes and Spirometry:

The Godart Pulmotest Model 1.A. 700 was used (see figure 1).



Godart Pulmotest

Figure 1

Functional Residual Capacity:

This was determined by employing the closed circuit helium technique.^{9,37} The patient was settled comfortably and was given time to adjust to the mouthpiece and the nose-clip. Readings were commenced at the end of a normal expiration and were monitored constantly. After a minimum interval of eight minutes, when three consecutive identical concentrations of helium were obtained, the point of equilibrium was said to exist.

Maximum Breathing Capacity:

This was measured by having the patient breathe maximally for 15 secs.¹² and multiplying the value so obtained by four to give the minute volume.

Expiratory Reserve Volume:

After a normal expiration the subject was asked to expire maximally. This was repeated until reproducible values were obtained (within 50 ml). The largest value was then recorded.

Residual Volume:

The residual volume was calculated by subtracting the expiratory reserve volume from the functional residual capacity.

Forced Vital Capacity:

This was measured by having the patient take a maximal inspiration followed immediately by a maximal forced

expiration. The total volume was the forced vital capacity. The test was repeated until reproducible values were obtained (within 50 ml.), and the largest value recorded. The volume obtained in the first second was termed the Forced Expiratory Volume, One Second, or FEV₁. Here too, reproducibility within 50 ml. was required, and the largest volume was recorded.

Diffusion Capacity:

Diffusing capacity was measured utilizing the steady state method.⁸ After breathing the mixture for two minutes, the end tidal pCO was then measured.

Normal Values:

The Cardiopulmonary Laboratory of the University of Alberta Hospital uses the values of Baldwin, Cournand and Richards.⁷ All gas volumes were measured at ambient temperature and pressure, saturated with water vapour and converted to body temperature and pressure saturated with water vapour.

MECHANICS OF BREATHING

Oesophageal Pressure:

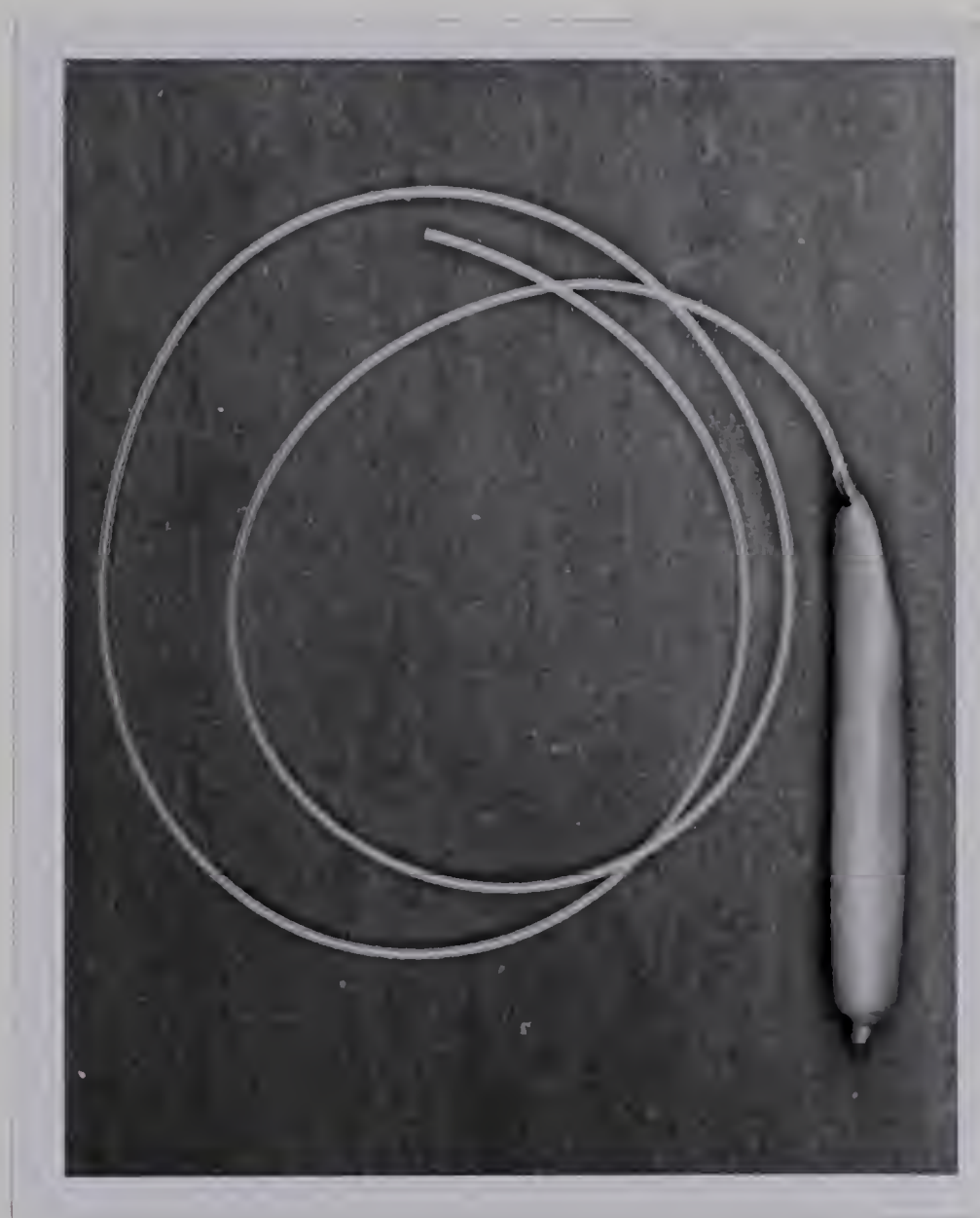
Oesophageal balloons were constructed after the design of Dr. J. Millic Emili, then of the Department of Physiology, Harvard School of Public Health.³⁸ These balloons are made of very thin latex, and are 10 cm long and 3 - 5 cms. in circumference (see figure 2). These are placed over the end of a polyethylene tube of 1 mm internal diameter in which

a series of hole have been drilled in a spiral pattern at 0.5 cm. intervals. The balloon covers this portion of the tube and is sealed to the tubing with Plioband^R adhesive. For the post operative measurements, the balloon was tethered by its very tip to a No. 16 French Levin Tube. (figure 3) All balloons were constructed either by the staff of the Inhalation Therapy Department of the University of Alberta Hospital or by the author himself.

For the post operative measurements the balloon was lubricated with K-Y^R jelly and passed into the subjects oesophagus via the nostril. In order to avoid the errors inherent in measurements taken from the upper or lower thirds of the oesophagus, the balloon was placed in the mid third of the oesophagus.

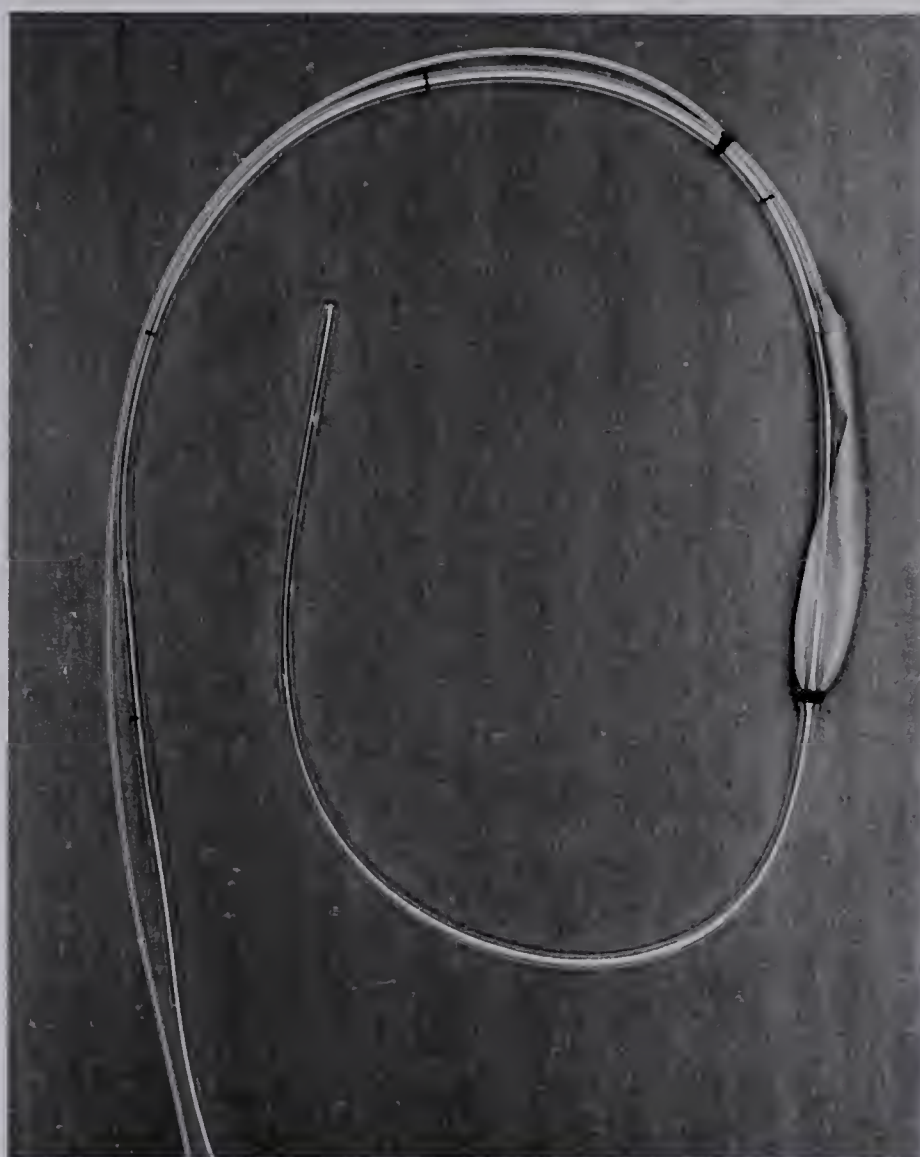
The distance was usually 37 - 43 cms from the external nares. When placed, the tube was adjusted to give the maximum pressure amplitude. The precise distance was carefully marked on the tube.

For the post operative measurements, the balloon was then attached to the Levin tube in such a manner that on insertion the balloon was in the same position in the oesophagus while the tip of the Levin tube was in the pyloric region of the stomach.



Oesophageal Balloon

Figure 2



Oesophageal Balloon with Levin Tube

Figure 3

After the balloon was inserted it was attached via the free end of the tubing to a Stathan differential Strain guage transducer. By performing a valsalva manoeuver, the subject emptied the balloon; which was then checked for integrity by injecting 5 ml of air and recovering it during a subsequent valsalva manoeuver. The balloon was then minimally inflated with 0.2 ml. of air in order to separate the walls. The system was calibrated against a water manometer at each reading.

Measurement of Flow Rate and Volume:

Flow rates and volumes were measured utilizing a Med-Science Electronics Inc. Wedge Spirometer Model 370 (see figure 4).

The Model 370 Wedge is a waterless, accurate and sensitive spirometer which provides electrical output proportional to volume and flow. It is specifically designed to produce a minimum loading of the patient and to reproduce the breathing pattern most faithfully.

The Wedge consists of two parallel square pans which are hinged to each other along one edge. One pan, containing a 2" inlet tube, is fixed to a stand and the other pan swings freely with respect to the fixed one. The space between the two pans is sealed air-tight with a vinyl bellows which is extremely flexible in the direction of pan motion, but offers high resistance to "ballooning" in or out of the spirometer,



Wedge Spirometer

Figure 4

i.e. a pressure difference between the Wedge interior and the atmosphere will cause negligible distortion of the bellows.

When gas is introduced into the Wedge, or withdrawn from it, the moving pan will change its position so as to compensate for the volume changes. The construction is such that the pan moves in response to very slight changes in volume. The instrument has been designed to offer low resistance to air flow (large inletport, low friction ball bearing hinges and highly flexible bellows) and to acceleration (small inertia). These features assure an undetectable dynamic loading on the patient's lungs. Under normal breathing conditions the pressure differential between the atmosphere and the interior of the Wedge is a fraction of a millimeter of water.

The static loading is of the same minimal order of magnitude, by virtue of the vertical mounting of the pans (a feature which could not be obtained with conventional, water-seal spirometers).

Volume and flow signals are obtained independently (neither integration nor differentiation steps are performed) from two linear transducers. These transducers are attached to the fixed frame and their cores are coupled to the edge of the moving pan. One transducer produces a DC signal proportional to displacement (volume), the other has a DC output proportional to velocity (flow).

The transducer outputs are connected by cable to the solid state electronic unit which may be mounted on the Wedge frame, in a standard relay rack, or placed inside a desk mounting cabinet for greater versatility. This electronics unit contains the power supply, an amplifier, the built-in calibration networks, balance and gain controls. It also contains switches for reversing the polarity of either or both volume and flow output signals and an impedance switch for matching the instrument to the recorder being used (either penmotor amplifier combination or a high sensitivity galvanometer type).

Calibration:

Calibration is available on both channels. A lever switch introduces the calibration signal and superimposes it on the existing signal. A selector switch determines the magnitude of the calibration signal. The volume may be calibrated with a signal corresponding to either 0.5, 1 or 5 litres. The flow calibration signals are factory adjusted to the particular Wedge by means of special fixtures. A volume of 1 litre is introduced in the one case, a flow rate of 1 litre per second in the other case, and the calibration signals are adjusted to produce equal signals. If a change in the magnitude of either calibration signal is desired, it may be changed by means of the slotted potentiometer mounted from the top of the chassis.

Special difficulty is encountered in checking the calibration of this apparatus. Volume can easily be scrutinized by comparing readings of a water spirometer. Flow however presents a different problem, since the small maximum capacity of the spirometer prevents the use of any other type of flow-meter. It was therefore decided that the best calibration could be done by checking the accuracy of the volume output and then manually integrating the recorded flow signal from the spirometer at both high and low flow rates and checking this against its own recorded volume signal. (see Appendix IV).

Recorders:

Two types of recorders were used. For the pre-operative measurements, an Electronics for Medicine Oscilloscope Recorder type DR 8 was used. This is a machine with a wide range of paper speeds (5-200 mm/sec) and time lines (0.004-1 sec.).

The Electronics for Medicine Oscilloscope Recorder type PR 7 was used for making the postoperative measurements. While not having the same vast range as its sister model the type DR 8, comparable speeds and time lines in the range used in this study can be obtained.

Preoperative time lines	0.04 sec.
paper speed	75 mm/sec.
Postoperative time lines	0.04 sec.
paper speed	25 mm/sec. and 75 mm/sec.

The recorders were set so that 1 mm volume deflection represented 20-35 ml. of air, 1 mm pressure deflection represented 0.2-0.35 cm. of water and 1 mm of flow deflection represented 2.8-3.5 and 1.04-1.4 litres/sec. in the preoperative and the postoperative measurements respectively.

PROCEDURE

Within the 48 hour period prior to surgery the subject was taken to the mechanics laboratory where an oesophageal balloon was introduced. The subject then breathed into the spirometer at 40-50 breaths per minute and 20-40 breaths were recorded. (This rate was selected in order to minimize the influence of cardiac impulses on the pressure tracing). The subject was allowed to select a comfortable tidal volume (range 530 ml-1670 ml) and encouraged to keep this constant.

Following the procedure the subject proceeded to the Cardiopulmonary Laboratory where routine pulmonary function studies were made.

On the morning of surgery the Levin Tube with attached oesophageal balloon was placed by the author. The subject was then taken to surgery. In all but one case (case #9*) 4-6 hours postoperatively the measurements of the

*Owing to the fact that the patient had respiratory depression, an endotracheal tube was placed for the first sixteen hours after surgery. Measurements were made at 18 hours after surgery.

mechanics of breathing were done in the recovery room. Since these patients could not be taxed, they were allowed to select their own breathing rate and tidal volume. The average tidal volume was about 2/3 the preoperative value and the average rate of respiration was 20-25 breaths per minute.

ANALYSIS OF THE RECORDS

Theory:

The act of breathing is one of moving a volume of air in and out of an elastic container through a conducting system of tubes. There will be, therefore three forces to overcome:

1. the elasticity of the system
2. the flow resistance of the system
3. the inertia of the system and the entity moved;
i.e. the air.

Elasticity is defined as the quantity of force necessary to cause a specific linear displacement. i.e. the elastance or stated in a formula:

$$E = \frac{\Delta F}{\Delta L}$$

The elastance is a quantity not generally used in respiratory physiology. The reciprocal of elastance, i.e. the compliance is the more universally described parameter:

$$C = \frac{\Delta L}{\Delta F}$$

However in respiratory physiology, the volume exchange is analogous to the linear displacement, and the pressure across the system is analogous to the force. Therefore the formula is restated.

$$C = \frac{\Delta V}{\Delta P} \quad \begin{array}{l} \text{(in litres)} \\ \text{(in cm.H}_2\text{O)} \end{array}$$

The pressure referred to in this study is the trans-pulmonary pressure. This is the gradient between the pressure at the airway opening and the intrapleural pressure. This is the vis a tergo causing volume exchange.

The intrapleural pressure is itself a resultant of two forces;-the elastic recoil of the lung, and its opposite vector, the elastic recoil of the chest wall. At rest this is a measureable quantity, and constitutes the restine intrapleural pressure. This is a negative quantity at rest and in inspiration, and in normal expiration is negative. However in a forced expiratory manoeuver, and/or when the airflow resistance is increased in expiration, it may become a positive quantity.

The oesophageal pressure used in this study; and taken as being an adequate measure of the intrapleural pressure. The validity of this assumption is supported by several workers.^{13,35}

In defining compliance in this study we have taken the resting intrapleural pressure to be the point of no flow

at end expiration. At peak inspiration, a point of no flow is again reached. The pressure difference between these is compared with the volume exchange to determine the compliance. This is in fact "dynamic compliance".^{1,33}

Using the principles outlined by Mead and Whittenberger³⁶ and by Ferris³¹ we may now determine the airflow resistance. During the act of volume exchange, there is an additional force to be overcome:- that of airflow resistance. At any one point in time, there will therefore be two partitions of the transpulmonary pressure which may be defined:- that pressure necessary to overcome the elastic recoil of the lungs, and that needed to overcome airflow resistance.

Resistance is defined by the physicist as the quantity of force necessary to cause a unit of flow; or as a formula:

$$R = \frac{\Delta F}{L \cdot \text{i.e. } (L/t)}$$

or in the terms of the physiologist:

$$R = \frac{\Delta P}{V \cdot \text{I.E. } (V/t)} \quad \begin{array}{l} \text{in Cm H}_2\text{O} \\ \text{in litres per second} \end{array}$$

Now remembering that the "elastic pressure" will always be in inverse proportion to the compliance we may separate the transpulmonary pressure into its two components, the "elastic pressure" and a residual pressure, the "dynamic pressure" which in effect is that causing airflow. Comparing

this latter with the flow rate we may then determine the resistance.

The resistance measured is the airflow resistance. This is composed of the active airway resistance and the frictional resistance in the tissues. The tissue resistance is a small proportion in the normal individual,³¹ (i.e. about 1/6 of the total airflow resistance).

The third opposing force is the inertia of the system. This is a minute quantity and is disregarded in this study.³⁴

The work of breathing is a measurement which can be made using but two variables, the pressure and the volume. The physicist defines work as a force moved a given distance; or

$$W = P \times V$$

Again in the terms of the respiratory physiologist;

$$W = P \times V$$

Using the transpulmonary pressure and volume records, and employing the method described by McIlroy, Marshall and Christie³² we may synthesize a figure which has several component parts as shown in figure 5.

In the diagram, the triangle ACD represents the work done in overcoming the elastic resistance of the lungs, and is termed the Elastic Work. The area ABCA represents that work necessary to overcome airflow resistance during

inspiration. During expiration energy is expended to overcome airflow resistance. A portion of this energy is derived from that stored as in the elastic tissues of the lungs during inspiration. This entity is represented by an area ACEA. The remaining energy is derived from the active work done by the muscles of expiration, and is represented by the area AEFA. This quantity is termed the Expiratory Resistive Work. The quantity CEDC is termed the negative work, and is said to represent work done by the muscles of inspiration which continued to contract during the early part of expiration.¹

TECHNIQUES

In this study 20 breaths were analyzed from each subject in both the preoperative and postoperative measurements. The breaths were analyzed consecutively unless an obvious artefact such as oesophageal spasm were superimposed on the pressure curve. This method was established as valid by Lynne-Davies.²⁸

Compliance:

Using the procedure outlined above compliance was measured and the mean was determined. It was expressed in litres per centimeter of water.

Resistance:

The mid-volume (taken as our standard) point was determined in each respiratory excursion and the parameters

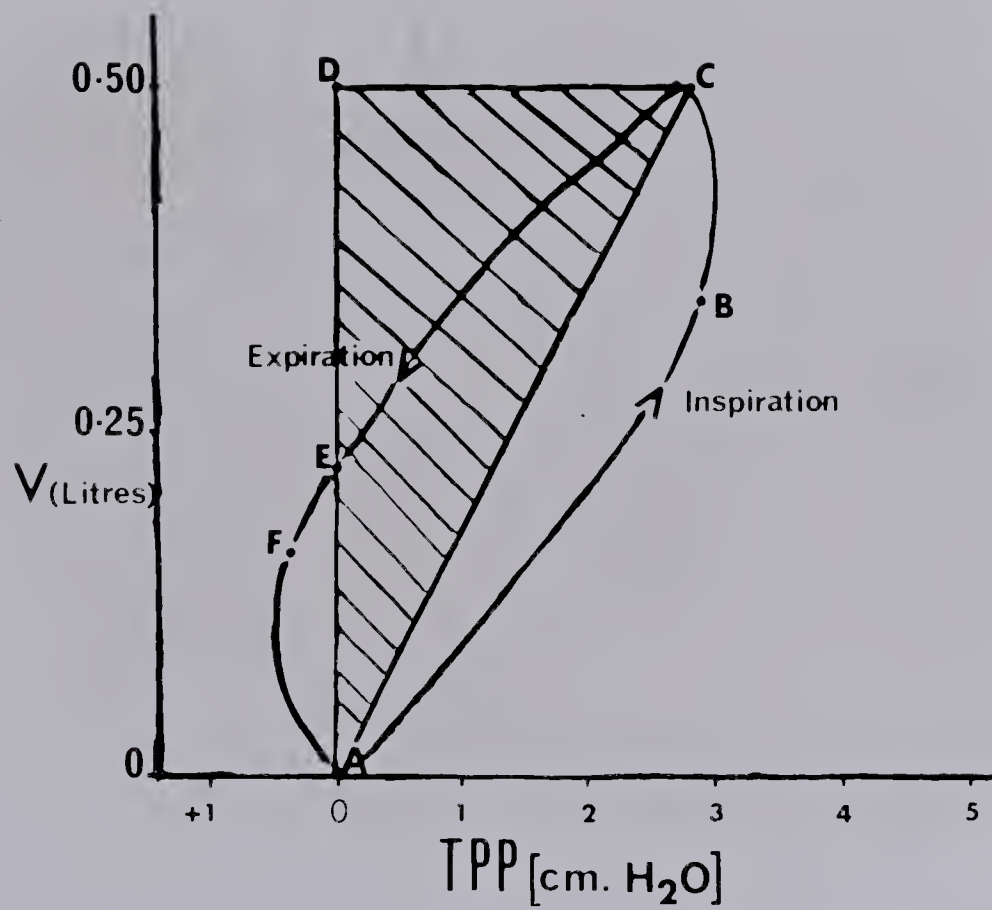


Diagram Of the Work Loop

Figure 5

were measured. The resistance was expressed in centimeters of water per litre per second.

Work of Breathing:

The pressure volume diagrams were constructed on graph paper ruled in one millimeter squares. Large scales were employed to lessen the errors in area measurements. The breath chosen for these diagrams was that most closely in agreement with the means of the compliance and resistance. A compensating polar planimeter was used to measure the areas.

In order to make a meaningful comparison of the work of breathing preoperatively and postoperatively the work of breathing per breath was calculated. Since rate and depth of respiration varied, measurement of the work per minute or the work per volume and attempting to compare these volumes preoperatively and postoperatively would be meaningless.

There is, however, a great difficulty inherent in making the comparison of the work of breathing per breath at two different rates and depths of respiration. The two can be compared if one of the breaths is taken as the standard and the other is converted mathematically to the same rate and depth of respiration.

Elastic Work:

The essence of the problem is to determine what the Elastic Work of breath #2 would be if it were taken at the same volume as breath # 1.

$$\text{Work} = PV$$

$$\text{"Elastic Pressure"} = \frac{1}{C} V$$

Now since the Elastic Work is represented in the pressure volume diagram as a triangle and the formula for the area of a triangle is

$$A = \frac{1}{2} b h \quad (\text{in our terms } a = \frac{PV}{2})$$

We may combine the formulae:

$$\text{Elastic Work} = \frac{1}{2} \frac{VV}{C}$$

$$\text{or} \quad \text{Elastic Work} = \frac{V^2}{2 C}$$

It must be remembered that in our conversion we do not wish to compensate for changes in compliance, since this is the cause of the variation in elastic work if the volume is constant. We must therefore eliminate it from the formula, and restore the relation: Elastic Work V^2

Now for breath # 1

$$EW_1 \propto (V_1)^2$$

and for breath # 2

$$EW_2 \propto (V_2)^2$$

To convert the elastic work at V2 to what it would be at V1

$$EW_2 \text{ (corrected)} \propto (V_1)^2$$

$$EW_2 \text{ (uncorrected)} \propto (V_2)^2$$

By cross multiplication

$$EW_2 \text{ (corrected)} = \frac{EW_2 \text{ (uncorrected)} (V_1)^2}{(V_2)^2}$$

Resistive Work:

In correcting the resistive work areas we must consider that the resistive work is proportional to two things if the resistance remains constant.

$$\text{Again} \quad W = PV$$

It is easily seen that if the volume increases, the work increases.

However, since we are considering the resistive work we must find that pressure due to the resistance. This is given in the formula:

$$P = R \dot{V} \quad \text{or} \quad P = R \frac{V}{t}$$

It is seen that as \dot{V} increases the pressure increases. Now we can combine the formulae:

$$\text{Resistive Work} = \frac{R \quad V \quad V}{t} \quad \text{or}$$

$$\text{Resistive Work} = \frac{R \quad V^2}{t}$$

As in our procedure with the Elastic Work, we do not want to compensate for changes in resistance; since, at constant volume and velocity, this is the function which determines the variation in work. Hence we eliminate it

from the formula:

$$R W \propto \frac{V^2}{t}$$

$$\text{For breath \# 1} \quad RW_1 \propto \frac{(V_1)^2}{t_1}$$

$$\text{For breath \# 2} \quad RW_2 \propto \frac{(V_2)^2}{t_2}$$

Again the problem is to correct the resistive work for breath # 2 to what it would be if it were taken at the same velocity and volume as breath # 1. Therefore

$$RW \text{ (corrected)} \propto \frac{(V_1)^2}{t_1}$$

$$RW \text{ (uncorrected)} \propto \frac{(V_2)^2}{t_2}$$

by cross multiplication

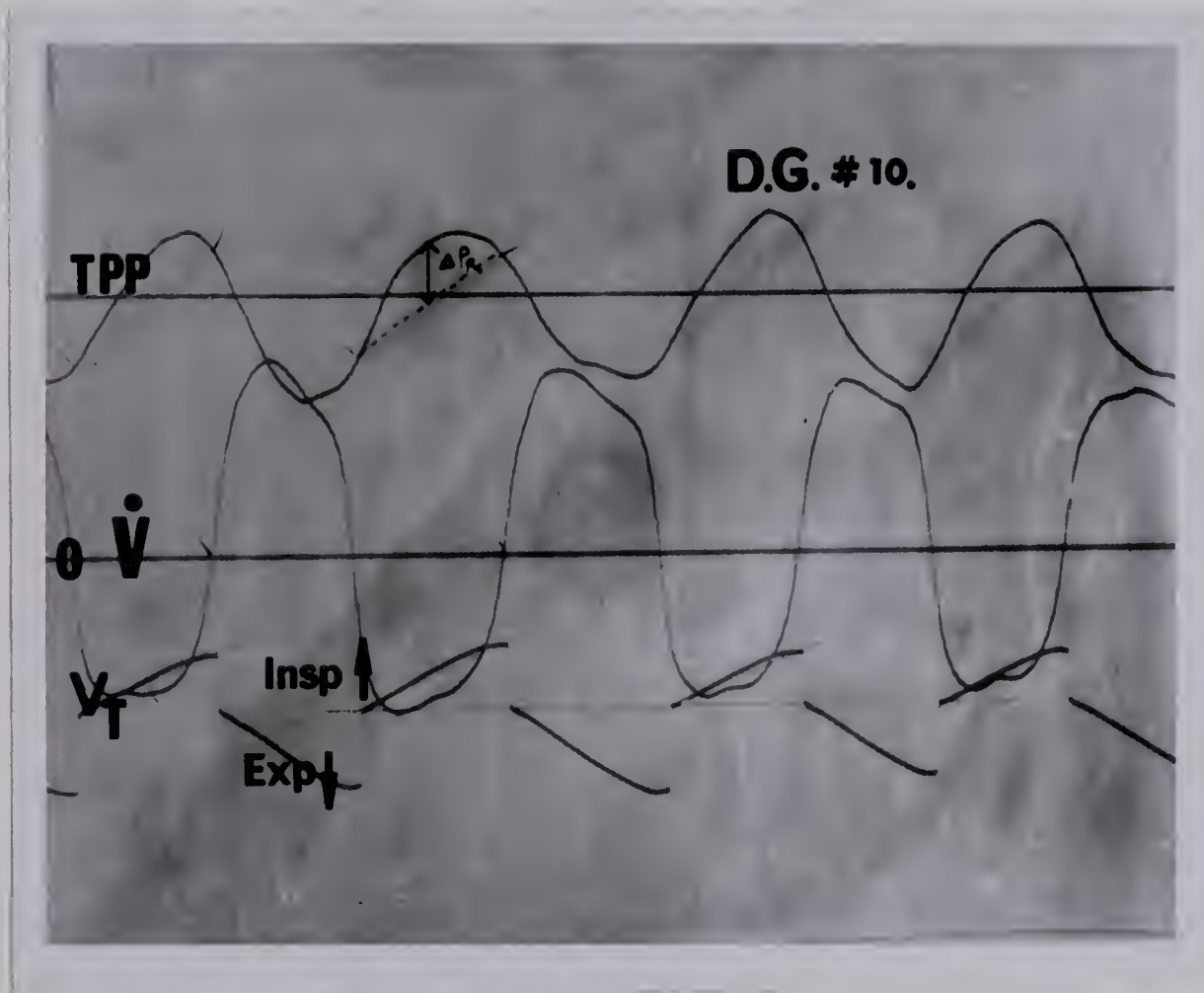
$$RW_2 \text{ (corrected)} = \frac{RW_2 \text{ (uncorrected)} (V_1)^2 t_2}{(V_2)^2 t_1}$$

In this manner, since we have corrected for variations in depth and rate of respiration we may now compare the work of breathing for a breath taken before and after any event or procedure which may have altered the remaining determinants of the work done, i.e. the compliance and resistance. 38

In this study the work of breathing is expressed in kilograms centimeters per breath.

STATISTICAL ANALYSIS OF RESULTS

The subjects served as their own controls. The preoperative and postoperative data were compared using the paired "t" test.^{3,29} The statistical tables used were those of Fisher and Yates¹⁸ and those of Arkin and Colton.²



Sample Tracing

Figure 6

RESULTS:

TABLE I

CASES STUDIED

Patient Number	Age	Height	Weight (lbs.)	Diagnosis	Sex
1	44	5'7½"	130	ASD	F
2	51	5'10¾"	139	MS, MI	F
3	22	5'5½"	117½	ASD	F
4	37	5'4"	157	MI	F
5	45	5'5¾"	160	ASD	F
6	50	5'5¼"	151	MI, AS (Minimal)	M
7	54	5'11"	217½	AS	M
8	26	5'9"	135½	MS, AI (Minimal)	M
9	47	5'9"	173	AS, AI, MS, MI, TS, TI	F
10	52	5'6"	158	MS, MI	M
11	50	5'1"	130	MS, MI, TS	F
12	51	5'11½"	193	AS, AI	M
13	46	5'11"	154	MS	M
14	19	5'4¾"	99 ¾	Tet.	F
15	28	5'7¼"	133¼	AI*	M
16	14	5'4"	148½	AS	M
17	63	5'4½"	127	MS, MI	F
18	38	5'4"	125½	AS	F
19	50	5'6"	133¼	AS, MI (functional)	M

ASD	=	atrial septal defect
AS	=	aortic stenosis
AI	=	aortic incompetence
MS	=	mitral stenosis
MI	=	mitral incompetence
TS	=	tricuspid stenosis
TI	=	tricuspid incompetence
Tet.	=	tetralogy of Fallot

* with rupture of sinus of valsalva into right atrium.

TABLE II

Patient Number	VC	%VC	FEV ₁	%	MBC	%	DLCO
1	2.36	60	1.98	85	63	72	12.9
2	4.42	102	3.11	70	90	95	14.8
3	3.74	101	2.92	78	76	76	32.7
4	3.19	90	2.28	71	120	138	14.8
5	3.27	89	2.29	70	76	93	14.9
6	3.63	88	2.06	57	97	97	4.8
7	5.61	121	3.83	68	172	164	10.9
8	5.11	101	4.08	80	139	109	30.3
9	2.45	61	1.59	65	58	69	9.2
10	4.69	113	3.09	66	97	98	12.6
11	2.13	70	1.51	71	76	105	8.2
12	5.07	105	2.42	48	66	61	20.1
13	4.22	84	3.63	72	99	79	24.9
14	2.30	62	2.01	87	80	78	12.7
15	4.79	100	3.20	67	100	81	17.0
16	3.49	82	3.01	86	108	98	15.0
17	2.52	75	1.91	75	44	65	11.7
18	3.35	96	2.77	83	107	124	20.5
19	2.63	63	1.50	57	77	76	8.3
Mean	3.63	87.5	2.59	71.3	91.3	93.6	15.83
SD	1.06	17.9	1.54	10.19	29.1	25.61	6.85

TABLE III

Patient Number	Time on Bypass (minutes)	Time of Anaesthesia (minutes)
1	32	225
2	51	235
3	38	175
4	62	265
5	41	105
6	86	240
7	110	225
8	36	240
9	234	440
10	73	285
11	191	320
12	80	295
13	114	322
14	88	300
15	168	380
16	151	350
17	45	185
18	85	280
19	95	270
Mean	91.05	220.26
SD	51.44	74.28

TABLE IV

COMPLIANCE

Patient Number	Preoperative	Postoperative	
1	0.133	0.063	
2	0.187	0.074	
3	0.111	0.151	
4	0.087	0.071	
5	0.078	0.064	
6	0.092	0.078	
7	0.226	0.180	
8	0.201	0.099	
9	0.174	0.119	
10	0.221	0.129	
11	0.232	0.081	
12	0.163	0.085	
13	0.209	0.193	
14	0.119	0.079	
15	0.211	0.237	
16	0.052	0.019	
17	0.135	0.090	
18	0.082	0.047	
19	0.119	0.082	
Mean	0.149	0.102	Mean Diff. -0.046
SD	0.056	0.052	0.045

TABLE V

INSPIRATORY RESISTANCE

Patient Number	Preoperative	Postoperative
1	2.08	6.53
2	1.34	0.76
3	1.85	0.96
4	1.85	2.38
5	1.96	2.14
6	2.21	3.39
7	0.95	1.19
8	1.11	0.59
9	1.23	1.13
10	2.76	1.58
11	1.55	2.24
12	1.63	1.88
13	0.73	0.85
14	1.00	1.25
15	3.01	1.94
16	0.99	0.78
17	0.99	1.14
18	1.90	2.35
19	3.22	4.10
Mean	1.703	Mean Diff. 1.956 .253
SD	0.705	1.406 1.169

TABLE VI
EXPIRATORY RESISTANCE

Patient Number	Preoperative	Postoperative	
1	2.24	13.43	
2	1.68	3.26	
3	2.36	4.08	
4	2.62	5.60	
5	2.48	4.65	
6	2.59	4.44	
7	1.62	1.76	
8	1.38	4.99	
9	1.97	2.86	
10	3.08	3.10	
11	2.16	6.92	
12	2.39	5.09	
13	1.60	3.50	
14	2.71	5.65	
15	3.88	1.56	
16	1.17	1.39	
17	1.63	3.52	
18	2.59	14.33	
19	2.16	6.92	
Mean	2.227	5.108	Mean Diff. 2.881
SD	.630	3.390	3.368

TABLE VII

MEAN RESISTANCE

Patient Number	Preoperative	Postoperative	
1	2.16	9.98	
2	1.51	2.01	
3	2.10	2.52	
4	2.24	3.99	
5	2.22	3.39	
6	2.40	3.92	
7	1.27	1.48	
8	1.25	2.78	
9	1.60	1.99	
10	2.92	2.34	
11	1.86	4.58	
12	2.01	3.49	
13	1.17	2.18	
14	1.86	3.45	
15	3.45	1.75	
16	1.09	1.08	
17	1.31	2.33	
18	2.24	8.34	
19	2.69	5.51	
Mean	1.966	3.506	Mean Diff. +1.540
SD	.619	2.227	2.138

TABLE VIII
ELASTIC WORK

Patient Number	Preoperative	Postoperative	
1	4.56	9.65	
2	1.51	3.79	
3	6.60	4.73	
4	12.63	15.50	
5	5.38	7.24	
6	9.85	9.40	
7	4.41	9.07	
8	4.51	9.16	
9	8.10	12.75	
10	3.89	6.81	
11	4.35	12.56	
12	2.32	3.83	
13	1.34	1.50	
14	4.99	8.33	
15	1.65	1.39	
16	7.55	12.15	
17	4.31	7.15	
18	5.65	8.26	
19	5.17	7.59	
Mean	5.196	8.098	Mean Diff. 2.902
SD	2.786	3.924	2.507

TABLE IX

INSPIRATORY RESISTIVE WORK

Patient Number	Preoperative	Postoperative	
1	4.76	20.35	
2	1.78	0.43	
3	5.43	1.68	
4	6.73	10.32	
5	2.48	4.57	
6	8.95	12.93	
7	3.61	3.83	
8	3.68	2.70	
9	4.48	4.43	
10	7.61	5.20	
11	2.86	4.21	
12	2.28	3.78	
13	0.26	0.50	
14	1.38	1.59	
15	4.17	3.29	
16	0.89	0.65	
17	1.17	1.56	
18	2.32	4.00	
19	6.42	7.83	
Mean	3.748	4.940	Mean Diff. 1.191
SD	2.358	4.826	3.851

TABLE X
EXPIRATORY RESISTIVE WORK

Patient Number	Preoperative	Postoperative	
1	1.38	23.76	
2	0.62	0.22	
3	1.27	0.56	
4	1.82	3.73	
5	0.61	1.72	
6	1.62	4.24	
7	2.08	0.12	
8	1.06	8.53	
9	2.79	4.48	
10	2.87	3.37	
11	1.34	2.35	
12	2.84	3.93	
13	0.14	1.47	
14	0.79	1.70	
15	4.13	1.23	
16	0.32	0.03	
17	0.42	2.75	
18	0.69	14.57	
19	2.78	6.15	
			Mean Diff.
Mean	1.559	4.469	2.910
SD	1.068	5.665	5.794

TABLE XI
NEGATIVE WORK

Patient Number	Preoperative	Postoperative
1	1.35	0.25
2	1.31	2.09
3	3.02	2.55
4	6.58	8.89
5	3.20	3.60
6	4.64	2.79
7	1.63	2.89
8	1.90	0.80
9	2.58	8.11
10	0.63	0.05
11	2.91	4.36
12	0.10	0.00
13	0.36	0.18
14	2.51	3.12
15	0.77	0.22
16	7.95	9.42
17	2.62	6.63
18	2.72	0.02
19	2.00	4.67
		Mean Diff.
Mean	2.568	3.192 0.624
SD	1.958	3.038 1.957

TABLE XII
TOTAL WORK

Patient Number	Preoperative	Postoperative	
1	12.05	54.01	
2	5.22	6.53	
3	16.32	9.52	
4	27.76	38.44	
5	11.67	17.13	
6	25.07	29.36	
7	11.73	15.91	
8	11.15	22.29	
9	17.95	29.77	
10	9.64	15.43	
11	11.51	23.48	
12	7.54	11.54	
13	2.10	3.65	
14	9.67	14.74	
15	10.68	6.13	
16	16.71	25.25	
17	8.52	18.09	
18	11.38	26.75	
19	16.37	26.24	
			Mean Diff.
Mean	12.792	20.751	7.959
SD	6.039	11.917	9.702

TABLE XIII

	Preoperative Mean	Postoperative Mean	"t" value	"p" value
Compliance	0.149	0.102	4.38	0.001*
Inspiratory Resistance	1.703	1.956	0.93	0.4 0.3
Expiratory Resistance	2.227	5.108	3.65	0.01* 0.001
Mean Resistance	1.966	3.506	3.06	0.01* 0.001
Elastic Work	5.196	8.098	4.93	0.001*
Inspiratory Resistive Work	3.748	4.940	1.32	0.30 0.20
Expiratory Resistive Work	1.559	4.469	2.12	0.05* 0.02
Negative Work	2.568	3.192	1.36	0.20 0.10
Total Work	12.792	20.751	3.49	0.01* 0.001
Compliance	0.149	0.143 (corrected for change in FRC)	0.42	0.7 0.6

* Statistically Significant

DISCUSSION

The results of the study are listed in tables II - XIII. They are summarized in table XIII; and the results of statistical analysis are appended. There was found to be a statistically significant decrease in pulmonary compliance, an increase in expiratory resistance, an increase in elastic work, an increase in expiratory resistance work and an increase in the total work of breathing per breath.

Compliance:

The preoperative mean compliance of 0.149 exhibited a statistically significant decrease to 0.102 following surgery. It must be noted that the preoperative mean compliance closely agrees with that reported by Woolf and Aguzzi (0.154)⁵⁶ for left heart valvular lesions. The pulmonary compliance could be changed by several mechanisms. First, changes in the lung tissues, i.e. inflammation of the pulmonary parenchyma (on the basis of any of the factors considered to cause the Post Perfusion Lung Syndrome) might well be expected to increase tissue turgor. Second, it has been found that atelectasis and/or obstruction of the small airways causes a decrease in cyanamic compliance.⁴⁸ Third an alteration in the surfactant lining the alveoli could easily alter pulmonary compliance.¹ Fourth, there is another factor which alters compliance, however, and that factor is lung volume. According to Comroe,¹² and Ebert,¹⁶ lung elasticity is better expressed by the quantity

termed "specific compliance" which is expressed by the formula:

$$SC = \frac{C_1}{FRC}$$

This quantity is constant in the individual, if the lung tissues remain unabtered. Thus if the lung volume increases the compliance decreases. If it decreases, the compliance increases.

It was not possible in the present study, to measure the FRC postoperatively. If we take the data of Howatt et al²⁴ and apply it to our data we see an interesting result. Howatt and his co-workers found that the postoperative FRC was 70.8% of the preoperative mean. If we take our preoperative FRC to be unity, and the postoperative FRC to be 70.8% of unity we see that our mean preoperative specific compliance would then be 0.149 while the postoperative mean specific compliance would be 0.143; and there would be no statistically significant difference between the two measurements. It must be remembered that Howatt et al attributed the decrease in FRC to the non-specific trauma to the chest wall and incisional pain. These factors may well be assumed to be in operation in the population studied. It would be hazardous to disregard these factors. The implication is that the decrease in compliance is attributable to mechanical factors, and that one cannot postulate an alteration of the pulmonary parenchyma or of the pulmonary surfactant, on the basis of this present study.

The observation that the compliance changes in the immediate postoperative period is contradictory to the data presented by Sullivan et al,⁴⁹ but it must be noted that they measured total respiratory compliance and that no conclusions about lung compliance can be drawn from their data, since the effects of trauma to the chest wall, which might alter its compliance are not documented. Mandelbaum and Giammona³⁰ described changes in compliance, but the lungs of their animals were artificially ventilated. Cartwright et al¹⁰ presented evidence that static inflation can prevent this compliance decrease. It must also be noted that the changes described by Mandelbaum and Giammona³⁰ are minimal after two hours of bypass and that the mean duration of cardiopulmonary bypass in our subjects was ninety-one minutes.

There remains but one possibility to be considered; that is that a decrease in compliance due to parenchymal or surfactant changes is masked by the improvement in pulmonary hemodynamics following definitive surgical correction of the cardiac lesion.

Since there have not been described any permanent changes in the pulmonary parenchyma following open heart surgery, it is not unreasonable to assume that any acute inflammatory changes would have resolved within the six months period following surgery. Similarly one may assume that

The first part of the paper is devoted to the study of the

properties of the function $f(x)$ defined by the

equation $f(x) = \int_0^x f(t) dt$ and to the

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any changes in surfactant have also resolved. This would leave only the cardiovascular factors changed, since according to Howatt et al²⁴ the major changes in lung volume tend to resolve by the eighth postoperative day and certainly by the fiftieth postoperative day as per the data of Weintraub et al.⁵⁴ Woolf and Aguzzi⁵⁶ in their six month follow up assessment of the mechanics of breathing following open heart surgery failed to find a significant change in compliance.

To summarize:- while it is apparent that a definite change in compliance has taken place, this change is fully explicable on the basis of a lowering of the FRC, and no evidence therefore exists of a change in the pulmonary parenchyma or the surfactant lining. It is not denied, however, that such changes could have taken place. There is merely insufficient evidence to postulate their existence.

Expiratory Resistance and Mean Resistance.

The mean preoperative Expiratory Resistance was 2.227 Cm H₂O/L/sec. as opposed to a mean postoperative Expiratory Resistance of 5.108 Cm H₂O/L/sec. The preoperative Mean Resistance was 1.966 Cm H₂O/L/sec. compared with a postoperative value of 3.506. The only comparable series in the literature is that of Woolf and Aguzzi⁵⁶ who publish figures for resistance which are considerably higher. They found a preoperative Mean Resistance of 7.76 Cm H₂O/L/sec.

No explanation can be given for this discrepancy. However, Woolf and Aguzzi⁵⁶ do not publish enough data concerning the cardiac status of their patients to permit comparison of their series with those in this study. The inspiratory resistance exhibited no significant change. The major portion of the significant change in mean resistance must be considered to be due to the change in expiratory resistance.

There are several possible mechanisms for a change in resistance. First, retention of and inspissation of secretions in the airways is universally recognized to exist following a major surgical assault of any nature.¹² This of course would particularly be true following thoracotomy with resultant limitation in respiratory excursions. Shimizu and Lewis⁴⁸ have explored this problem. They concluded that the mucus plugs in the larger airways were accompanied by an increase in airflow resistance. They state that mucus plugs in the bronchioles caused a decrease in compliance. Second, mucosal edema can cause a decrease in the radius of the lumen of the airways. This mucosal edema could be caused by any of several mechanisms;- i.e. irritation by anaesthetic gases, inflammatory changes (as a result of any of the many factors discussed above in exploring the Post Perfusion Lung

Syndrome) or venous congestion. Third is spasm of the smooth muscles of the bronchi. This latter has not been described as a complication following open heart surgery. A fourth factor is purely mechanical. As lung volume decreases, so does the airway diameter. This subject has been extensively explored by Fry and Hyatt.¹⁹

The observation that expiratory resistance was significantly increased whereas inspiratory resistance failed to show significant increase is explained that the bronchial lumen is smaller during expiration and larger in inspiration.¹⁹ This would tend to magnify, during the expiratory phase, any factor increasing airflow resistance. The last factor cited will be explored first.

We have already conceded that the FRC has probably been significantly reduced in our patients during the immediate postoperative period. In order to quantitate the increase in resistance which we might expect on the basis of this decrease in lung volume, we may utilize the formula of Brisco et al as quoted by Comroe.¹²

$$R = \frac{4.2}{V} \quad \text{where } V \text{ equals the volume at which resistance is measured.}$$

Since in this study, airflow resistance is measured at $\frac{1}{2}$ tidal volume and postoperatively we noted a reduction of approximately 33% in the tidal volume we may then combine

this quantity with the decrease in FRC documented by Howatt et al.²⁵ Thus we may postulate a decrease in the lung volume of 66 - 70% at the time resistance is measured in the post-operative state. We may then postulate an increase in airway resistance of 44-50% in the basis of decreased lung volume alone. This would then give us a corrected value of 3.52 cm H₂O/L/sec. for postoperative mean expiratory resistance as opposed to a preoperative value of 2.22. The mean resistance would have a postoperative value of 2.46 as opposed to the mean preoperative value of 1.966. It is obvious that the magnitude of the differences are still such that another factor is probably operating.

The third factor cited, that of bronchospasm has already been discounted. Mucosal edema, the second factor may exist. The principal inflammatory changes described in connection with the Post Perfusion Lung Syndrome manifest themselves more in the bronchioles rather than in the larger airways. Shimizu and Lewis⁴⁸ present histological evidence indicating that obstruction in the smaller airways manifests itself as a compliance change, rather than as a change in airflow resistance. This would tend to obviate any of the factors which produce the Post Perfusion Lung Syndrome as operating to

produce the increase in expiratory resistance (and therefore in mean resistance) seen here.

The anaesthetic (Halothane) used in all our patients is not described as having irritant properties; indeed by direct action, it is a bronchodilator.²¹ This factor cannot therefore be held responsible.

The first factor alone remains, i.e. retention of secretions in the airways. This was posited to be responsible for the increase in postoperative airflow resistance noted by Shimizu and Lewis.⁴⁸ According to their beliefs, it is mucus retained in the larger airways, bronchi and trachea which produce the increased resistance.

It is interesting to note in this regard, that one of the patients who showed the greatest increase in resistance had a previous history of bronchial disease, which would tend to favour the retention of secretions.

The Work of Breathing:

The changes noted in various portions of the work of breathing are rather easily accounted for on the basis of the foregoing discussion of the changes noted in their analogous measurements of compliance and resistance.

Elastic work increased parallel with the decrease in compliance. This is solely a reflection of the compliance change because the postoperative measurements are corrected

to the preoperative tidal volumes.

Similarly the expiratory resistive work was increased paralleling and attributable to the increase in the expiratory resistance.

The total work of breathing increased as a result of an increase in all its divisions, but principally as a result of the increases in elastic work and expiratory resistive work.

It must be emphasized that the postoperative values represent, not the actual work done per breath in the postoperative state but the work done were the breath to be taken under precisely the same conditions as in the preoperative state, i.e. at the same rate and depth.

CONCLUSIONS

On the basis of this study, the following conclusions may be made:

There is, during the first postoperative day a decrease in pulmonary compliance. The decrease is not thought to be due to any alteration in the lungs; but, is thought to be due to the fact that the lungs are operating at a lowered volume.

There is, during the first postoperative day an increase in both expiratory airflow resistance and in mean airflow resistance. This increase is thought to be due,

in part, to the fact that the lungs are operating at a lowered volume and therefore airway diameter is reduced. The retention of secretions in the airways is thought to play a concomitant role in producing the increase in airflow resistance.

There are, during the first postoperative day, increases in elastic work, expiratory resistive work and the total work of breathing per breath which are attributable to the decrease in compliance in the case of elastic work, and to the increased expiratory airflow resistance in the case of expiratory resistive work, and to both in the case of the total work of breathing per breath.

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APPENDIX I - CASE SUMMARIES

Case #1

This is a 44 year old white female who was admitted with a diagnosis of atrial septal defect. The diagnosis was made incidental to physical examination. Her past history included many respiratory tract infections attributed to bronchiectasis which was treated by lobectomy nine years previous to this admission.

Under cardiopulmonary bypass the atrial septal defect was repaired. Two days following surgery the patient began to have difficulty in clearing bronchial secretions. This condition remained for several days. The postoperative course was otherwise essentially uneventful.

Case #2

This 38 year old white female was admitted for repair of her mitral valve. For twelve years prior to admission she had symptoms of fatigue and dyspnea.

Clinical and laboratory examinations were compatible with a diagnosis of mitral stenosis and incompetence.

Utilizing cardiopulmonary bypass a cutter valve prosthesis was inserted. Aside from a slightly lowered systemic blood pressure during the early postoperative period, the postoperative course was unremarkable.

Case #3

This is a 22 year old white female who was admitted for repair of an atrial septal defect. She had a past history of rheumatic fever at age 15. The clinical and laboratory examinations revealed a large atrial septal defect.

The defect was repaired utilizing cardiopulmonary bypass. The postoperative course was uneventful.

Case #4

At age fourteen, this thirty seven year old white female was first discovered to have rheumatic heart disease. The clinical and laboratory findings were diagnostic of mitral incompetence.

A Cutter prosthetic valve was inserted using cardiopulmonary bypass. No postoperative complications were noted.

Case #5

Twenty six years prior to admission this forty-five year old white female was noted to have a heart murmur. She had a history of vertigo with exercise. Clinical and laboratory examination confirmed a diagnosis of atrial septal defect.

Under cardiopulmonary bypass a preicardial patch was used to repair the defect. The postoperative course was uneventful.

Case #6

This fifty year old white male was admitted with symptoms of a left sided valvular lesion of one years duration. There was no previous history of rheumatic fever. Clinical and laboratory examination confirmed a diagnosis of mitral incompetence and minimal aortic stenosis.

During cardiopulmonary bypass it was adjudged that the dominant lesion was aortic stenosis and that the mitral incompetence was secondary. A Storr-Edwards Valve was inserted in the site of the aortic valve.

The postoperative course was uneventful.

Case #7

This fifty four year old white male was admitted with a history of angina, dyspnea and syncope, all of three years duration. He had had a myocardial infarction six years prior to this admission. Clinical and laboratory examination confirmed a diagnosis of aortic stenosis.

Under cardiopulmonary bypass a Storr-Edwards Prosthetic Valve was placed in the site of the aortic valve. The postoperative course was uneventful.

Case #8

This patient is a twenty-six year old white male who first became aware of fatigue and dyspnea some five years prior to admission. There was no previous history suggestive of rheumatic fever.

The clinical and laboratory examinations led to preoperative diagnosis of mitral stenosis and aortic incompetence.

Using cardiopulmonary bypass the orifice of the mitral valve was dilated. It was felt the aortic incompetence was minimal. The patient made an uneventful recovery.

Case #9

This forty-seven year old white female had a history of attacks of rheumatic fever at ages six and twenty. Symptoms of a left sided valvular lesion did not appear until age thirty-six. These symptoms became progressively worse until the time of admission. The clinical and laboratory examinations led to a diagnosis of stenosis and incompetence of the aortic, mitral and tricuspid valves.

Under cardiopulmonary bypass the defective valves were replaced. For the first twelve hours after operation the patient was ventilated with a positive pressure respirator using an oropharyngeal airway. This was done because of a respiratory depression

Case # 10

This fifty-two year old white male had no previous history suggestive of cardiac disease until three months prior to admission when he had a sudden onset of dyspnea on exertion.

The clinical and laboratory examinations confirmed a diagnosis of mitral stenosis and incompetence.

Using extracorporeal circulation a Kay-Shirley prosthesis was inserted in the site of the mitral valve. The postoperative course was uneventful.

Case #11

This is a fifty year old white female who was admitted with a diagnosis of mitral stenosis and incompetence and tricuspid stenosis. She had a previous history of rheumatic fever. She had experienced symptoms of a left sided valvular lesion for ten years prior to this admission. Clinical and laboratory examination confirmed the admitting diagnosis.

Utilizing extracorporeal circulation the mitral and tricuspid valves were replaced. The postoperative course was marred by cardiac arrhythmias which were noted for five days after surgery.

Case #12

This is a fifty-one year old white male who was admitted for repair of an aortic valve lesion. The clinical and laboratory findings were compatible with a diagnosis of aortic stenosis and incompetence.

Using cardiopulmonary bypass the aortic valve was replaced with a Storr-Edwards prosthesis. The immediate postoperative course was uneventful.

Case #13

This forty-six year old white male had had rheumatic fever at age sixteen and twenty-five. He was asymptomatic until one year prior to admission when he began to experience easy fatigue and dyspnea.

The clinical and laboratory examinations were compatible with a diagnosis of mitral stenosis.

A cutter valve prosthesis was inserted in place of the mitral valve. This procedure was carried out using cardiopulmonary bypass. The postoperative course was unremarkable.

Case #14

At age seven, this thirty-seven year old white female had an operation to repair a Tetralogy of Fallot. For the four years prior to this admission she had experienced dyspnea, syncope and parasthesis of the face and tongue.

Clinical and laboratory examination led to the impression that the original repair had been inadequate.

Under cardiopulmonary bypass the intraventricular septal defect was repaired and the right ventricular outflow tract was enlarged. The postoperative course was not marred by any complications.

Case #15

This twenty year old white male had been shot in the chest with a 22 cal. rifle bullet seven years before this admission. Following this wound he developed aortic insufficiency.

The clinical and laboratory examination confirmed the diagnosis.

While under cardiopulmonary bypass the aortic valve was replaced with a cutter prosthesis and a fistula leading from the sinus of valsalva into the right atrium was repaired. There were no postoperative complications.

Case #16

This thirty-six year old white male was admitted with a history of one episode of vertigo. There was no previous history of heart disease.

Clinical and laboratory tests confirmed a diagnosis of aortic stenosis.

Using cardiopulmonary bypass the aortic valve was replaced with a cutter prosthesis. The postoperative course was unremarkable.

Case #17

This a sixty-three year old white female who was admitted with a history of rheumatic fever at age 15 and several embolic episodes over the subsequent years.

For several years prior to admission she had experienced dyspnea on exertion.

Clinical and laboratory examination confirmed a diagnosis of mitral stenosis and incompetence.

The defective valve was replaced with a cutter prosthesis using cardiopulmonary bypass. The postoperative course was unremarkable.

Case #18

This thirty-eight year old white female had several bouts of rheumatic fever between ages six and twenty-nine. Since her second pregnancy thirteen years before this admission, she had been under treatment for her symptoms of peripheral edema and dyspnea on exertion.

The clinical and laboratory findings established a diagnosis of aortic stenosis.

The aortic valve was replaced with a cutter prosthesis utilizing extracorporeal circulation.

The postoperative course was unremarkable.

Case #19

This fifty year old white male was admitted with a diagnosis of aortic stenosis with incompetence of the mitral valve. He had had rheumatic fever as a youth. For two years prior to admission he had experienced exertional dyspnea.

The defective aortic valve was replaced with a Storr-Edwards prosthesis. The procedure was carried out using cardiopulmonary bypass. At the time of operation it was felt that the mitral incompetence was secondary to the aortic lesion. The postoperative course was uneventful.

APPENDIX II

NORMAL SUBJECTS

Five normal subjects were studied. The following results were obtained.

Subject Number	Compliance	Inspiratory Resistance	Expiratory Resistance
1	0.144	1.55	2.65
2	0.330	1.38	1.36
3	0.128	2.18	2.75
4	0.245	0.99	1.09
5	0.097	1.69	2.89
Means	0.189	1.56	2.15

APPENDIX III

Effect of Esophageal Balloon Being Attached to Nasogastric Tube.

One subject was studied. The esophageal balloon was placed in the esophagus and the pulmonary mechanics were determined. The balloon was then removed and tethered to the Levine tube. It was then replaced in the esophagus at the same depth and the mechanics of breathing determined at one-half hour, and at one hour. The results were as follows:

	Compliance	Inspiratory Resistance	Expiratory Resistance
Balloon	0.175	1.83	2.15
Balloon + Levine Tube			
$\frac{1}{2}$ hour	0.181	1.76	2.21
1 hour	0.166	1.85	2.23

These figures are well in accord with the variations within a normal subject reported by Lynne-Davies.

APPENDIX IV

CALIBRATION OF WEDGE SPIROMETER

To calibrate the volume output of the Wedge spirometer it was coupled to the Godart Pulmotest I.A. 700. When a weight was placed on the bell of the pulmotest, the volume was displaced into the wedge spirometer. The table below gives the comparative figures.

Pulmotest	Wedge Spirometer	% Error
5065	5061.0	0.8%
1035	1037.1	0.2%
510	510.6	0.8%

To calibrate the flow output, it was recorded simultaneously with the volume output and then manually integrated.

Volume	Flow Integrated to Volume	% Error
3020.5	3016.8	1.3%
1540.7	1541.2	0.3%
325.2	324.9	0.9%

In addition, an attempt was made to determine the internal resistance of the wedge spirometer. A pressure lead was taken from the interior of the wedge bellows.

This pressure lead was attached to the Statham strain guage used in these studies and amplified using the Electronics for Medicine PR7 recorder. The amplification was such that a pressure of one centimeter of water would give a displacement of fifteen millimeters on the recorded tracing.

A subject was then asked to pant as rapidly as possible into the wedge spirometer. No pressure change could be detected in the spirometer during this manoeuver.

SPECIFICATIONS

Operating Volume - 10 litres

Linearity - 0.5%

Volume Discrimination - 3 ml.

Base Line Stability - Negligible drift

Calibration Stability - 1%

Noise - 1 mV

Leakage - Zero

Resistance to Breathing - 0.2 mm H₂O pressure at normal
breathing levels

Static Pressure - 1 mm H₂O maximum

Dead Space of inlet port - 150 ml.

Residual Volume - 5 litres

Acceleration Loading - approximately 3 mm H₂O pressure at an
acceleration of 1000² litres per second
per second.

Frequency Response - Flat within 5% to 22 cps.

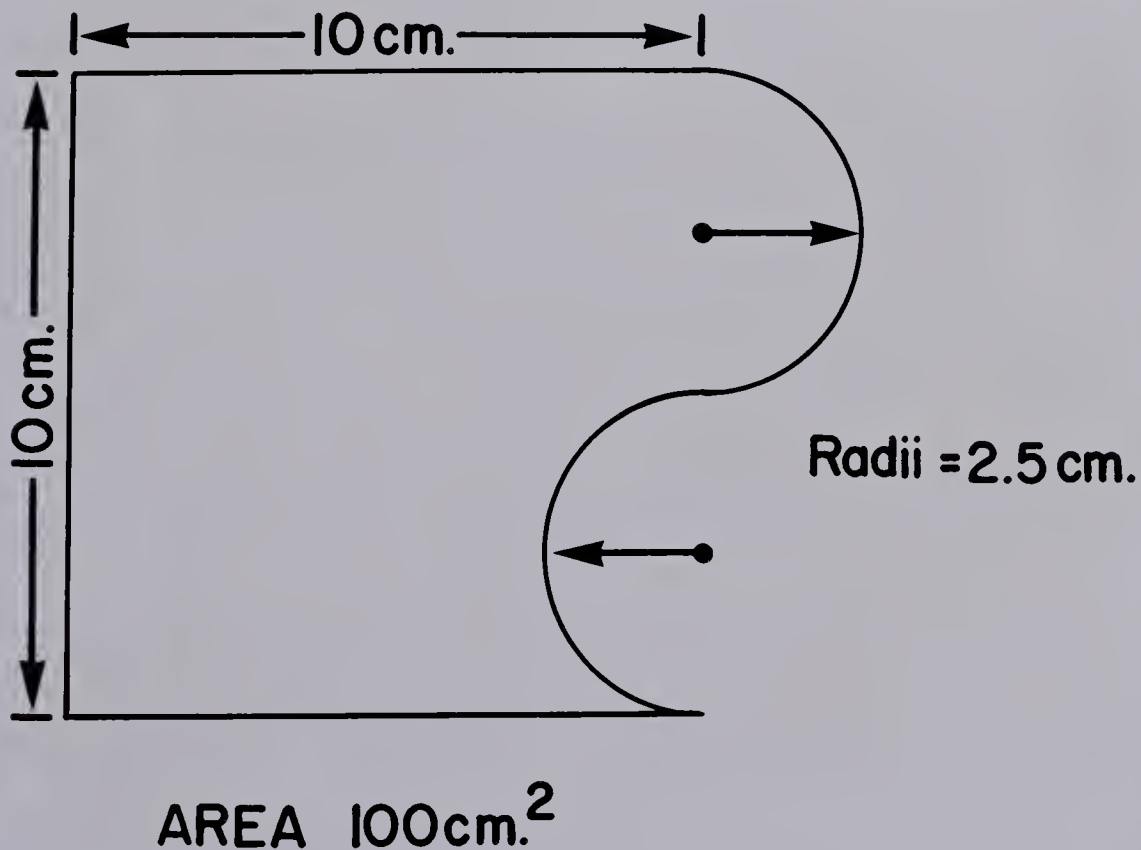
APPENDIX V

CALIBRATION OF POLAR PLANIMETER

The compensatory polar planimeter is a device for measuring the area enclosed by a two dimensional figure. This requires a manual operation. The operator, as well as the device itself must be calibrated. A figure with a complex outline, the area of which was 100 CM^2 was drawn and the area estimated three times.

The results are:

	Area estimated
	100.8
	100.5
	100.6
Mean	100.63
% Error	0.63%



Standare Figure for Polar Planimeter

Figure 7

APPENDIX VI

ABBREVIATIONS

A	=	area
B	=	base
C	=	compliance
DLCO	=	Diffusion capacity of carbon monoxide
E	=	elasticity
ER	=	expiratory resistive
ERW	=	expiratory resistive work
EW	=	elastic work
F	=	force
FEV ₁	=	forced expiratory volume, one second
FRC	=	functional residual capacity
h	=	height
IR	=	inspiratory resistance
IRW	=	inspiratory resistive work
L	=	linear displacement
L [•]	=	linear displacement/unit time
MBC	=	maximal breathing capacity
%MBC	=	maximal breathing capacity expressed as % of predicted normal value
MR	=	Mean resistance
p	=	pressure
pCO	=	partial pressure carbon monoxide
R	=	resistance

RW	=	resistive work
SC	=	specific compliance
t	=	time
T.P.P.	=	transpulmonary pressure
VC	=	vital capacity
%VC	=	vital capacity expressed as percent of predicted normal value
W	=	work

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